Effects of positive pressure ventilation on cardiovascular physiology

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This chapter addresses section F10(i) of the 2023 CICM Primary Syllabus, *"Describe the physiological consequences of intermittent positive pressure ventilation and positive end-expiratory pressure"*. This chapter specifically addresses the cardiovascular effects which were asked about in Question 20 from the second paper of 2019. In short, positive pressure ventilation affects preload, afterload and ventricular compliance, and the effect in most situations is a decrease in cardiac output. However, the effect may be beneficial in the context of decompensated heart failure, where the decreased preload and afterload result in a return to a more productive part of the Starling curve. In this rests the chief benefit of CPAP in the management of acute pulmonary oedema.

In summary:

Mechanisms affecting the right ventricle and the pulmonary circulation are:

- Increased intrathoracic pressure is transmitted to cenral veins and the right atrium, decreasing right ventricular preload
- Increased intrathoracic pressure is transmitted to pulmonary arteries
- Transmitted alveolar pressure increases pulmonary vascular resistance
- Increased pulmonary vascular resistance increases right ventriular afterload
- Thus, increased afterload and decreased preload has the net effect of decreasing the right ventricular stroke volume.

Mechanisms affecting the left ventricle and the systemic circulation are:

- Decreased preload by virtue of lower pulmonary venous pressure
- Decreased afterload due to a reduction in LV transmural pressure and an increased pressure gradient between the intrathoracic aorta and the extrathoracic systemic circuit
- Thus, the LV stroke volume improves in a failing ventricle, and declines in a healthy one.

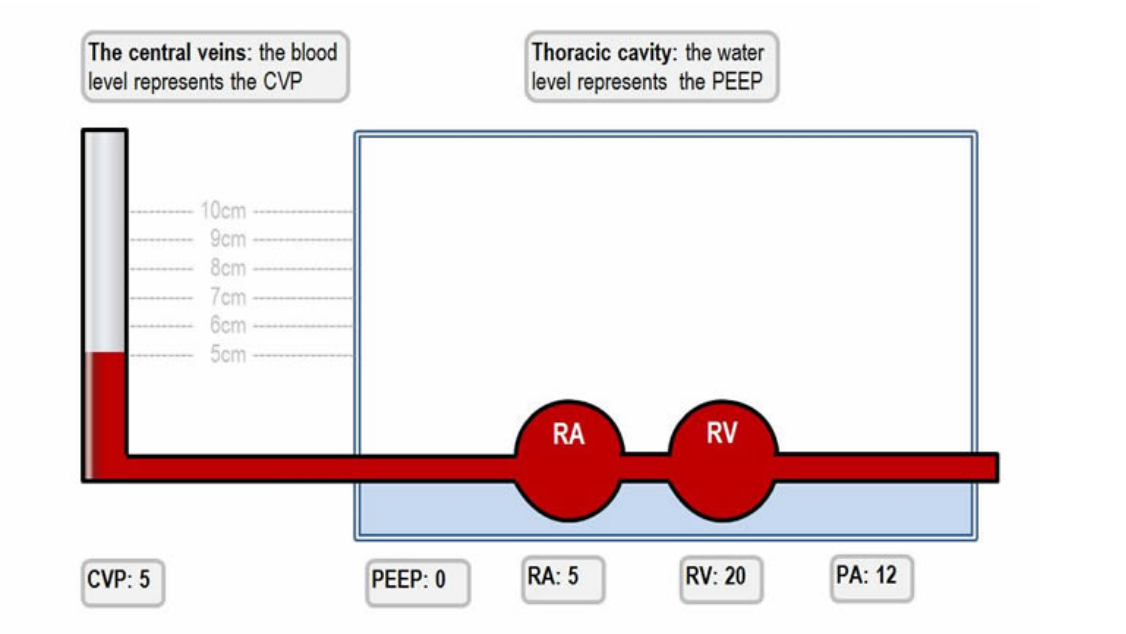
The consequences of this are:

- Decreased cardiac output
- Decreased myocardial oxygen consumption

Classical articles on this subject are Cassidy et al (1979) and Luce (1984), but these are paywalled by the powers that be. Something more recent, and more importantly free, is the article by Duke (1999), made available to all by CICM.

Effect of PEEP on preload

Here is a crude hydrodynamic representation of the intrathoracic circulation (well, everything up to the pulmonary veins, anyway). The pressures are all in cm H_2O to aid comparison with respiratory pressure, even though conventionally the pressure of blood is measured in millimetres of mercury.

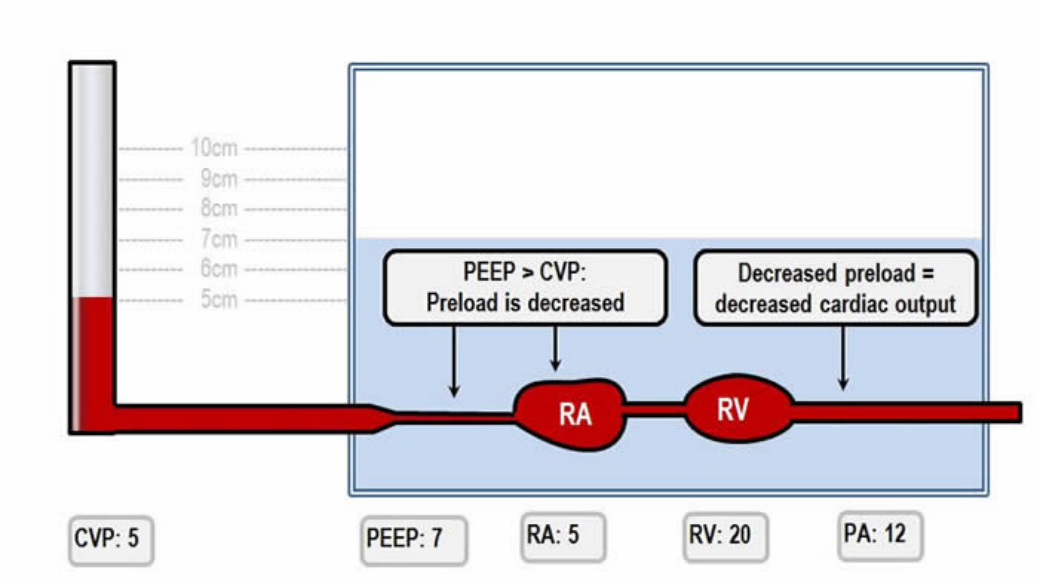


PEEP is essentially end-expiratory positive intrathoracic pressure. This influences the return of venous blood to the heart.

In a patient who is slightly underfilled, venous return is still adequate in the absence of PEEP, because the CVP is still higher than the (mostly) negative thoracic pressure.

Indeed, with inspiration, that pressure becomes even more negative, and blood is actively sucked into the chest.

Now lets add 7 cmH_2O of PEEP.



The PEEP is higher than the CVP, and venous return is impaired. On echo, the right atrium and IVC will appear collapsed.

This decreased preload will result in a decrease in cardiac output.

Obviously, one can adjust for this effect by increasing the CVP; another words, you can give your patient fluids and expect a decrease in positive pressure related hemodynamic effects.

Conversely, you can make use of this as a diagnostic tool, and surmise that the patient in whom PEEP produces hemodynamic instabilty is an underfilled patient.

These are oversimplified diagrams. In fact, PEEP is not transmitted directly to the venous system. In a lung with normal compliance, no more than 25% of the PEEP is transmitted to the central veins. But however small, there is still an effect. And of course nobody in the ICU has normally compliant lungs…

Effect of PEEP on right ventricular afterload

The effects of PEEP on RV afterload can be summarised as "it increases". If we decide to conceptualise afterload as wall stress, then the relationship of right ventricular parameters can be interpreted in Laplacian terms, as the function of

the product of volume and pressure:

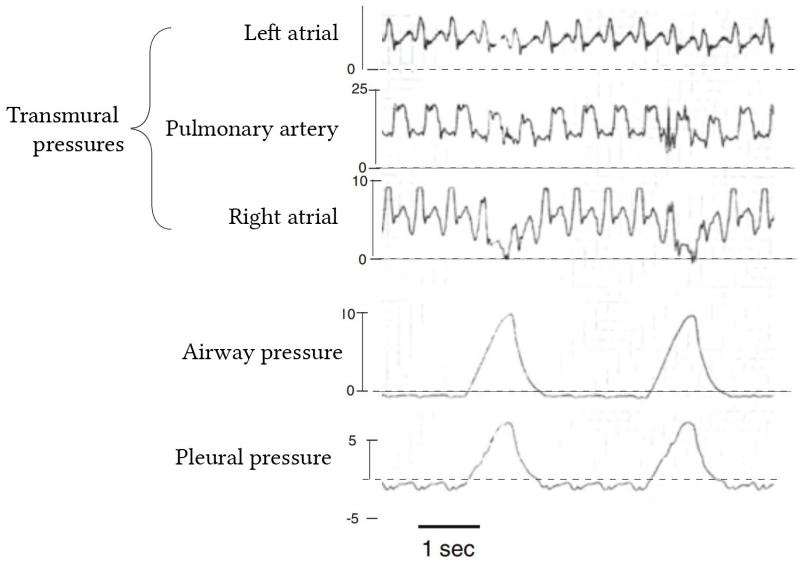
$$\sigma \propto Pr/T$$

where

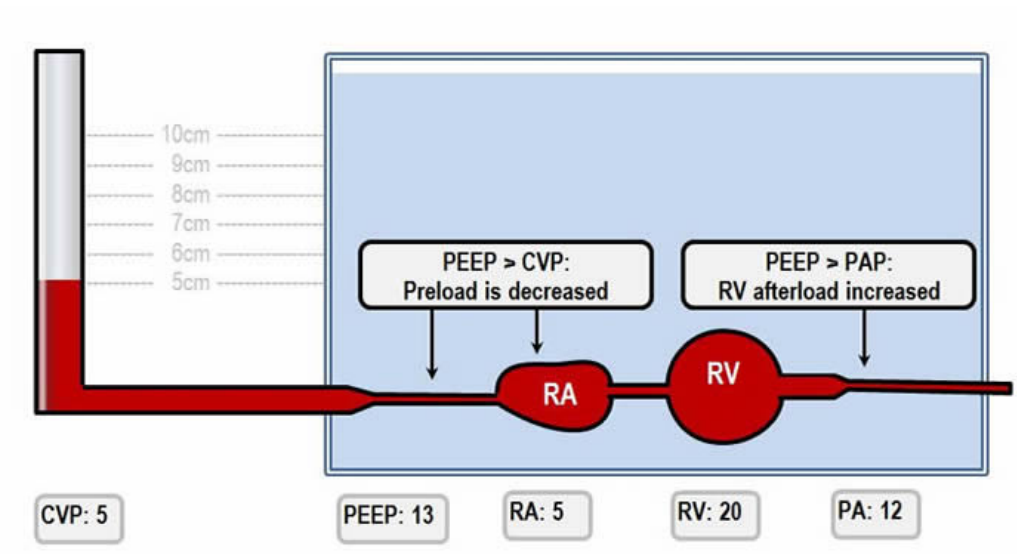
- σ is the wall stress
- P is the Laplace pressure, or the pressure difference between the inside and the outside of the sphere (i.e. the ventricular transmural pressure, which is the difference between the ventricular cavity pressure and the intrathoracic pressure)
- r is the radius of the ventricle
- T is the thickness

The right ventricle is far from spherical in shape, but we forgive ourselves the inaccurate physics in order to empower the explanation. Luecke et al (2005) and Tedford (2014) do it better, of course, but the reader is not here for the official version of events. In summary, for all intents and purposes the Laplacian behaviour of the RV is mostly determined by the systolic pressure, as the radius and thickness remain relatively stable outside of insane events. RV wall stress is therefore mostly proportional to the pressure generated during systole, i.e. the pulmonary arterial systolic pressure. This, in turn, is affected by PEEP to a variable degree:

- Positive alveolar pressure is transmitted to the pulmonary arteries, increasing pulmonary vascular resistance. Cheifetz et al (1998) demonstrated this experimentally in cute baby piglets, where PVR basically doubled when the tidal volumes were increased from 10 to 20ml/kg.
- Positive pressure recruits collapsed alveoli, decreasing the pulmonary vascular resistance that might have been elevated by mechanical collapse and hypoxic vasoconstriction.
- Pulmonary artery transmural pressure (i.e. intravascular pressure relative to pleural) decreases, as does the transmural pressure of all the atrial chambers, as in this recording from Mesquida et al (2011):



But do we need to discuss transmural pressures in every chamber? Yes, a huge amount of complexity is lost by explaining things in this fashion (what of the arterial wave reflections, inertance of blood, compliance of the pulmonary circulation, etc), but for the intents and purposes of this discussion, all these will be reduced to pulmonary vascular resistance. The optimal pulmonary vascular resistance is at FRC, so the degree to which intrathoracic pressure increases the pulmonary vascular resistance depends to a considerable extent on how far from FRC one ends up, i.e. profoundly overdistended and profoundly underinflated ventilated lungs will increase the pulmonary vascular resistance, whereas optimally recruited lungs will return it to some kind of minimum.



Additionally, most of the positive intrathoracic pressure is transmitted directly to the RV, at least in animal models where Morgant al (1965) found a difference no greater than 2mmHg between he pleural and pericardial pressures. This should reduce the RV transmural pressure, which is the difference between the pressure inside and outside the RV wall: if the RV chamber pressure was 20 mmHg and PEEP was 10 mHg, the transmural pressure would be (20 - 10) = 10 mmHg.

Lastly, it would be wrong to completely dismiss the effects of radius and wall thickness. Preload is reduced by PEEP, as noted above, which also brings down pressures across the entire pulmonary circulation, and reduces the radius of the RV overall. On the other hand, the increased pulmonary arterial pressures being about an end to the systolic ejection at a higher end-systolic volume.

Thus, in summary, the net effect of intrathoracic pressure on right ventricular afterload therefore depends on two opposing influences:

- Increased afterload (increased wall stress) due to:
    - Increased pulmonary arterial pressure, due to deviation from optimal pulmonary arterial resistance at FRC
    - Higher end-systolic RV volume due to increased PA pressure
    - Thus, increased RV end-systolic radius, and therefore greater Laplacian wall stress
- Reduced afterload (reduced wall stress) due to:
    - Reduced end-diastolic volume (effect of lower preload)
    - Reduced transmural pressure due to direct transmission of intrathoracic pressure to the cardiac chambers
    - Reduced radius and increased thickness due to lower preload

The net effect of this on any given ventricle is difficult to guess, which is reflected in the variability of experimental data, such as the findings by Jardin et al (1991), that *"in some patients a given level of PEEP improved lung mechanics and reduced RV size, while in other patients the same level of PEEP worsened lung mechanics and caused RV enlargement".*

Effect of PEEP on left ventricular compliance

Left ventricular compliance is decreased by a leftward shift of the interventricular septum. PEEP, by causing a dilatation of the right ventricle (as a result of increased right ventricular afterload) supposedly causes the interventricular septum to bulge into the left ventricle during diastole (because the LV end-diastolic pressure ends up being less than the RV end-diastolic pressure).

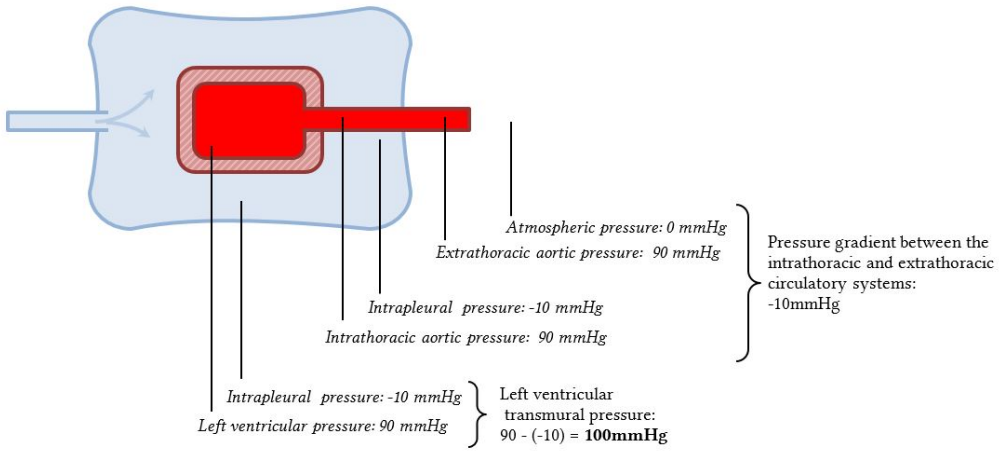
As a result, left ventricular preload is decreased even more, and cardiac output decreases.

Effect of PEEP on left ventricular afterload

Spontaneous ventilation is surprisingly unfavourable for LV workload, given that it is the "natural" state of affairs. However, if we view LV afterload as "all of the factors that contribute to total myocardial wall stress (or tension) during systolic ejection", we observe that the negative pressure generated during a spontaneous breath is actually a disadvantage for the left ventricle.

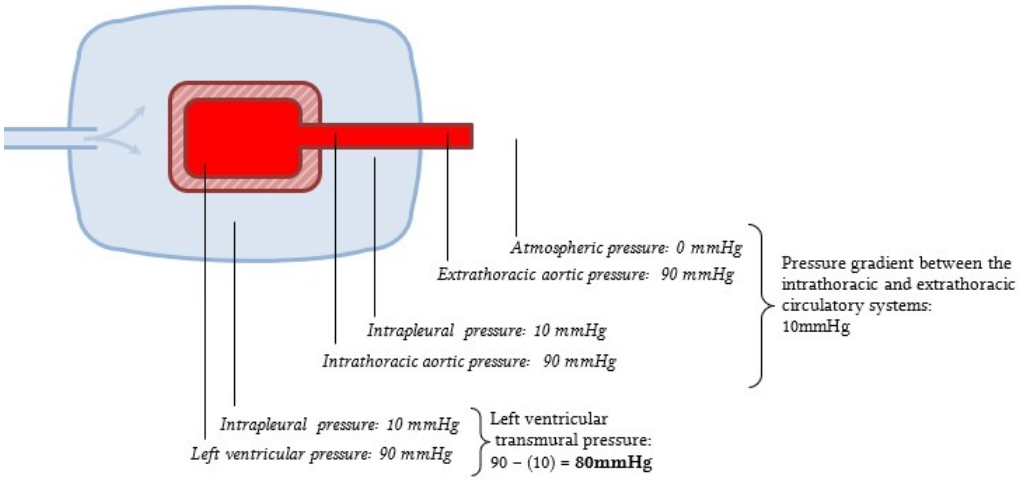
In order to discuss this, we may separate afterload into the LV transmural pressure (which describes the wall tension of the LV, and therefore afterload) and the pressure differences between the intrathoracic and extrathoracic vascular compartments.

The healthy spontaneously breathing patient with good lung compliance and normal tidal volumes generates a negative pressure in the realm of 1-2 mmHg, which is miniscule compared to the pressures generated by the mighty left ventricle (given that normally one must generate a pressure of around 80 mmHg just to open the aortic valve). The effect of increased LV preload due to spontaneous inspiration is in fact far more important, and tends to *increase* the stroke volume. The tiny increase in the afterload is lost in the noise, and the net effect is still positive.



This diagram exaggerates the spontaneous respiratory effort by tenfold in order to make the calculations more impressive, but this is unintentionally demonstrating what occurs in (for example) acute pulmonary oedema. Decreased lung compliance demands more negative pressures to achieve the same tidal volume, with disastrous effects on the LV transmural pressure. In this instance, the difference between intra-LV pressure and intrapleural pressure increases the LV transmural pressure, and therefore the afterload is also increased.

Positive pressure ventilation reverses this problem. Positive intrapleural pressure *decreases* LV transmural pressure (it is subtracted from the intra-LV pressure), with the resulting improvement in LV wall stress, oxygen consumption, and other favourable survival-improving things.



Additionally, putting positive pressure on the intrathoracic aorta causes a pressure gradient to develop between this vascular compartment and the extrathoracic circulatory system (which is under atmospheric pressure). The

resulting gradient favours the flow of blood into the systemic circulation, also decreasing LV workload.

So, given that the influence of intrathoracic pressure on LV afterload and aortic pressure gradients is so small, which actually contributes to the afterload reduction?

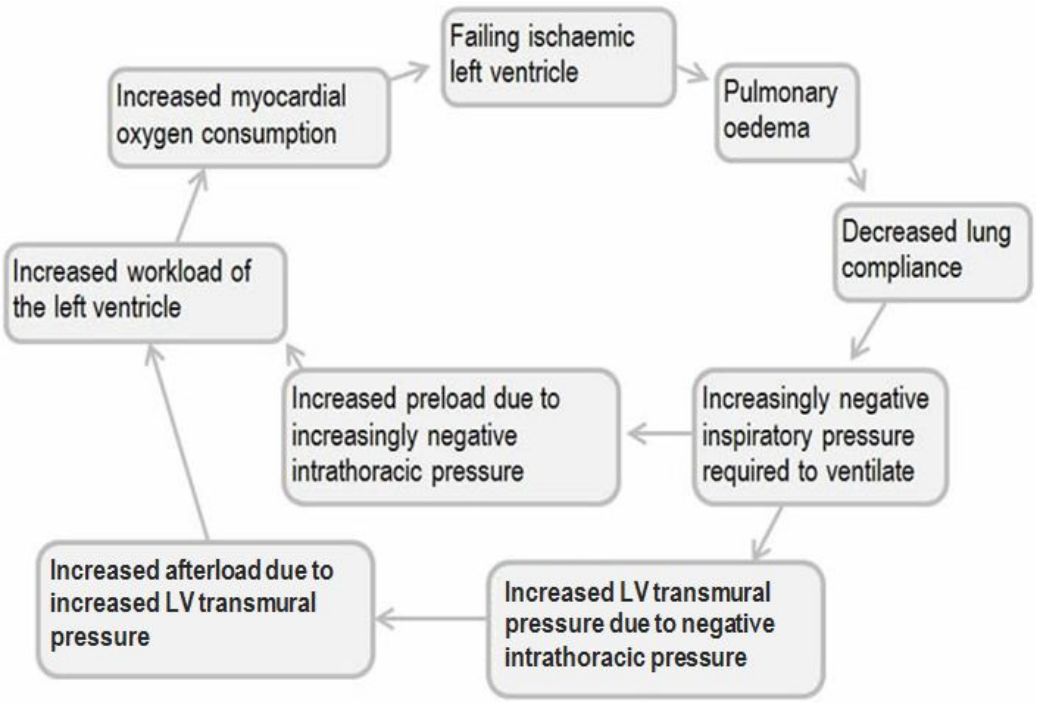
The best answer I have found is probably from Luecke, Thomas, and Paolo Pelosi ("Clinical review: positive end-expiratory pressure and cardiac output"). They write:

> "*In contrast to its effect on the right ventricle, PEEP has been shown to decrease LV afterload. PEEP increases the pressure around structures in the thorax and, to a lesser extent, in the abdominal cavity, relative to atmospheric pressure. Because the rest of the circulation is at atmospheric pressure, this results in a pressure differential, with most of the systemic circulation being under lower pressure than the left ventricle and the thoracic aorta. Thus, increased ITP, at constant arterial pressure, decreases the force necessary to eject blood from the left ventricle in a manner exactly analogous to decreased arterial pressure, at constant ITP* "

The paragraph then goes on to discuss the difficulties in defining and measuring LV transmural pressure, particularly how it would vary in the presence of a dilated myocardium. The authors conclude that "*it is difficult to assess whether the PEEP-induced afterload reduction is actually due to a reduction in LV end-systolic transmural pressure or simply related to the commonly observed decrease in mean arterial pressure.*"

Now, this updated section owes its existence to Ben, who correctly pointed out some rage-inducing errors in my previous interpretation of LV transmural pressure. I tried to thank him directly, but he left an inaccurate email address, and therefore Address resolution of uoguelpg.ca. failed: Domain name not found.

Anyway. This is the sequence of events which causes left ventricular failure to spiral out of control.



That's right. the left ventricular failure causes pulmonary oedema, which causes decreased lung compliance, which induces a greater respiratory effort, which in turn causes more and more negative intrathoracic pressure (as the patient gasps for air) and thus causes increased preload, increased afterload, and thus greater demand on the already failing left ventricle, which then decompensates further.

PEEP puts an end to this cycle by fighting the increasingly negative inspiratory pressure. The end-expiratory pressure increase decreases the preload and afterload, decreasing the demands on the left ventricle, and pushing it into the more efficient part of the Starling curve.

Previous chapter: Effects of positive pressure ventilation on pulmonary physiology

Next chapter: Haemodynamic changes during a mechanical breath

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